Risk Factors for Anterior Disc Displacement with Reduction and Intermittent Locking in Adolescents

Stanimira I. Kalaykova, DDS, PhD
Assistant Professor
Department of Oral Kinesiology

Frank Lobbezoo, DDS, PhD
Professor
Department of Oral Kinesiology

Machiel Naeije, PhD
Professor and Chair
Department of Oral Kinesiology

Academic Center for Dentistry Amsterdam (ACTA)
Research Institute MOVE
University of Amsterdam and
VU University Amsterdam
The Netherlands

Correspondence to:
Stanimira I. Kalaykova
Department of Oral Kinesiology
Academic Center for Dentistry Amsterdam (ACTA)
Gustav Mahlerlaan 3004
1081 LA Amsterdam
The Netherlands
Fax: +31-20-5980414
Email: s.kalaykova@acta.nl

Aims: To test the hypothesis that oral parafunctions and symptomatic temporomandibulair joint (TMJ) hypermobility are risk factors in adolescents for both anterior disc displacement with reduction (ADDR) and intermittent locking. ***Methods:*** *Participants were two hundred sixty 12- to 16-year-old adolescents (52.3% female) visiting a university clinic for regular dental care. ADDR and symptomatic TMJ hypermobility were diagnosed using a structured clinical examination. During the anamnesis, reports of intermittent locking and of several parafunctions were noted, eg, nocturnal tooth grinding, diurnal jaw clenching, gum chewing, nail biting, lip and/or cheek biting, and biting on objects. The adolescents' dentitions were examined for opposing matching toothwear facets as signs of tooth grinding. Risk factors for ADDR and intermittent locking were first assessed using univariate logistic regression and then entered into a stepwise backward multiple model.* ***Results:*** *While in the multiple model, ADDR was weakly associated only with increasing age (*P = .02*, explained variance 8.1%), intermittent locking was weakly correlated to diurnal jaw clenching (*P = .05*, explained variance 27.3%).* ***Conclusion:*** *In adolescence, diurnal clenching may be a risk factor for intermittent locking while age may be a risk factor for ADDR. Symptomatic TMJ hypermobility seems to be unrelated to either ADDR or to intermittent locking.* J OROFAC PAIN 2011;25:153–160

Key words: anterior disc displacement, human, internal derangement, oral parafunctions, symptomatic TMJ hypermobility, temporomandibular joint

Anterior disc displacement with reduction (ADDR) is the most common temporomandibular joint (TMJ) internal derangement encountered in adults and in children.[1] In a TMJ with an ADDR, in the closed mouth position, the articular disc is located anteriorly to the condyle. During mouth opening, the disc reduces by slipping back on top of the condyle, while at the end of closing, it again becomes anteriorly dislocated.[2]

ADDR is usually pain free and manifests itself by reciprocal clicking joint sounds at the moments of disc reduction and dislocation.[3] However, ADDR can sometimes develop into a more serious condition, as disc reduction may become hampered and may thereby cause a mouth-opening limitation.[2] The loss of reducing capacity can be temporary because some subjects with an ADDR report periods of sudden, transient, and often painful movement limitations (intermittent locking).[4–6] Reports of intermittent locking have been found predictive of the permanent loss of disc reduction (an anterior disc displacement without reduction, ADDWoR).[4,7] The latter condition is only rarely accompanied by signs of locking,[7] and, if such signs are present, these generally subside in time.[8,9]

Table 1 Number of Participants, Age, and Gender Distribution in the Study Sample			
Age (y)	N	Male (%)	Female (%)
12	49	29 (59.2)	20 (40.8)
13	54	27 (50)	27 (50)
14	54	19 (35.9)	35 (64.1)
15	57	27 (47.4)	30 (52.6)
16	46	22 (47.8)	24 (52.2)
Total	260	124 (47.7)	136 (52.3)

The etiology of ADDR may include several risk factors such as microtraumatic events resulting from oral parafunctions, hypermobility, and degeneration of the TMJ.[10] So far, however, the role of parafunctions in ADDR development is unclear because most studies have explored parafunctional activities in relation to clicking TMJ sounds[11–13] that are not only characteristic for ADDR, but also for other internal derangements.[14] Furthermore, while systemic hypermobility has been related to ADDR,[15] it is as yet unclear whether local symptomatic TMJ hypermobility (ie, characterized by jerky jaw movements and clicking joint sounds when the condyle snaps over the apex of the eminence) has a role in the onset of ADDR. Moreover, TMJ degeneration is not likely related to ADDR development because ADDR prevalence was found to increase in children, especially in adolescence, and not in adulthood, which would be expected if degeneration was involved.[1,16] This finding indicates that the risk factors for ADDR are most active during adolescence and, therefore, should be sought in that age period.

As yet, intermittent locking has not been studied thoroughly, and data on its risk factors and prevalence is scarce. In a recent experimental study, it was found that loading of the TMJ by means of an intensive chewing task may temporarily hamper disc reduction in subjects reporting intermittent locking.[6] This finding suggests that joint loading during oral parafunctions may contribute to intermittent locking.

Therefore, the aim of this study was to test the hypothesis that oral parafunctions and symptomatic TMJ hypermobility are risk factors in adolescents for both ADDR and intermittent locking.

Materials and Methods

Participants

The study sample was comprised of 260 adolescents, aged 12 to 16 years, who consecutively visited the students' clinic at the Academic Center for Dentistry Amsterdam (ACTA) in the period from September 2006 to March 2007 for regular dental care. The adolescents and their parents gave informed consent prior to participating; only one 16-year-old boy refused participation. The mean age of the sample was 14.0 ± 1.4 years, and 52.3% were female. The numbers of 12-year-old through 16-year-old participants were similar, and the female-to-male ratios were balanced (Table 1). All participants spoke Dutch fluently. This study was reviewed and approved by the Medical Ethics Committee of the VU University, Amsterdam, The Netherlands.

Clinical Examination

During the anamnesis, a structured checklist was used in order to document the adolescents' awareness of intermittent locking (ie, transient periods of sudden inability to open the mouth completely, during which joint clicking disappears), previous trauma to the orofacial area (eg, a blow to the lower jaw or TMJ area), and parafunctional activities (Table 2). The parafunctional activities included diurnal jaw clenching, gum chewing, nail biting, lip and/or cheek biting, and biting on objects (pens, pencils, etc). The adolescents were also asked whether they performed tooth grinding at night, or were told so by their family or friends. For all questions, the participants had to choose between possible answers "yes" or "no." The participants were questioned in a standardized way and, depending on the level of their maturation, questions were sometimes additionally explained in order to improve understanding.

During the physical examination, the participants' dentitions were inspected for the presence or absence of opposing matching tooth-wear facets, which are considered an intraoral sign of tooth grinding.[17] The amount of active maximum mouth opening was measured. The presence of joint clicking was observed by means of palpation while each participant was performing the following jaw movement tasks: maximal open-close movements, maximal opening and loaded closing movements (ie, with a manually applied, downward directed force of about 30N on the chin), and maximal open-close movements from a protruded jaw position (eg, from the incisal end-to-end position; elimination test).[18] There were at least three trials per task.

An ADDR was diagnosed on the basis of the following clinical criteria,[14,18,19] modified from the Research Diagnostic Criteria for TMD (RDC/TMD)[20]: *(1)* clicking on opening and (loaded) closing, reproducible on at least two of three consecutive trials and *(2)* elimination of clicking on open-close movements from a protruded jaw position.

Symptomatic TMJ hypermobility was diagnosed on the basis of the following clinical criteria:[18, 19] *(1)* clicking in the late part of opening and early part of (loaded) closing, in combination with jerky jaw movements, reproducible on at least two of three consecutive trials and *(2)* no elimination of clicking on open-close movements from a protruded jaw position.

Statistical Analyses

The prevalence of ADDR and reported intermittent locking in at least one of the joints was calculated for the study sample. Risk factors for these internal derangements were assessed using logistic regression analyses. In the analyses, TMJ diagnoses were used per subject, irrespective of the unilateral or bilateral character of the diagnoses.

The independent variables for the outcome variable "ADDR" were age, gender, history of trauma, reported parafunctions, observed matching tooth-wear facets, and symptomatic TMJ hypermobility. Participants without an ADDR diagnosis at the time of examination but reporting to have experienced intermittent locking were excluded from analysis in order to avoid inclusion of cases with ADDWoR as healthy. Univariate logistic regression analyses were used to examine which independent variables were significantly associated with the outcome variable ($P \leq .05$). Subsequently, the significant variables were entered into a stepwise backward logistic multiple regression model (P value to remove = .05) to identify the subset of independent variables that was best associated with the outcome variable. Odds ratios (OR) and their confidence intervals (CI) were obtained.

The independent variables for the outcome variable "reported intermittent locking" were the same as for "ADDR." Healthy participants (ie, subjects without an ADDR at the time of examination and not reporting intermittent locking) were excluded from the analysis as the loss of disc reduction is inherent to the presence of a disc displacement. The logistic regression analysis for the outcome variable "reported intermittent locking" was performed as described above.

Furthermore, logistic regression analysis was performed as described above in order to test the association of the presence of opposing matching tooth-wear facets with reported nocturnal tooth grinding, corrected for other parafunctions, gender, and age. All analyses were carried out with SPSS for Windows, version 16.0 (SPSS, IBM).

Table 2 Questions Posed to the Participants During the Oral History

1.	Have you ever experienced brief periods during which your jaw would suddenly get locked, so that your mouth couldn't open completely and your jaw would stop producing clicking sounds?	yes / no
2.	Have you ever had a trauma to your face, such as a blow to the jaw or the area of the joint?	yes / no
3.	Do you grind your teeth at night, or were you told so by your family members or friends?	yes / no
4.	Do you have the habit of clenching your jaws during the daytime?	yes / no
5.	Do you have the habit of chewing gum?	yes / no
6.	Do you have the habit of nail-biting?	yes / no
7.	Do you have the habit of lip- and/or cheek-biting?	yes / no
8.	Do you have the habit of biting on objects, such as pens or pencils?	yes / no

Results

The prevalence of ADDR and reported intermittent locking in at least one of the joints in the sample was 5% (7.9% for the girls, 2.5% for the boys; 13 subjects in total) and 6.2% (9.6% for the girls, 2.5% for the boys; 16 subjects in total), respectively. Of the adolescents diagnosed with an ADDR, 46.2% reported intermittent locking (ie, 2.3% of the study sample, or 6 subjects). From the children without an ADDR diagnosis, 4.1% reported to have experienced intermittent locking (ie, 3.9% of the study sample, or 10 subjects). ADDR and intermittent locking were unilateral, except for one 15-year-old female who had a bilateral ADDR.

The univariate logistic regression analysis indicated that age and symptomatic TMJ hypermobility were associated with the presence of an ADDR (Table 3). In the multiple logistic regression analysis, only age was associated with the presence of an ADDR (OR = 1.8, CI 1.1–3.0, P = .02, explained variance 8.1%).

The logistic regression analysis indicated already at the univariate level that, from all variables, reported intermittent locking was associated with reported diurnal jaw clenching (OR = 10, CI 1.0–104.5, P = .05, explained variance 27.3%; Table 4). The univariate analyses for reported intermittent locking in relation to reported nocturnal grinding and gum chewing could not be processed because of low counts of subjects reporting grinding and high counts of subjects reporting gum chewing, respectively (Table 4). However, the distribution of these counts over the subgroups of

Table 3 Univariate and Multiple Logistic Regression Analysis of Risk Factors for ADDR in at Least One of the TMJs

		Univariate analysis			Multiple analysis		
Variable	Count (%)	*P*	OR	95% CI	*P*	OR	95% CI
Age (continuous, in years)		.02	1.814	1.117–2.946	.02	1.814	1.117–2.946
Gender							
Boys	123 (49.2)		Reference		—	—	—
Girls	127 (50.8)	.07	3.448	0.926–12.847			
History of trauma							
No	141 (56.4)		Reference		—	—	—
Yes	109 (43.6)	.71	0.807	0.256–2.54			
Reported parafunctions							
Nocturnal tooth grinding							
No	216 (86.4)		Reference		—	—	—
Yes	34 (13.6)	.53	0.513	0.064–4.074			
Diurnal clenching							
No	172 (68.8)		Reference		—	—	—
Yes	78 (31.2)	.57	1.396	0.441–4.412			
Gum chewing							
No	14 (5.6)		Reference		—	—	—
Yes	236 (94.4)	.74	0.7	0.084–5.8			
Nail biting							
No	113 (45.2)		Reference		—	—	—
Yes	137 (54.8)	.11	2.91	0.781–10.843			
Lip and/or cheek biting							
No	139 (55.6)		Reference		—	—	—
Yes	111 (44.4)	.47	1.506	0.491–4.619			
Biting on objects							
No	155 (62)		Reference		—	—	—
Yes	95 (38)	.96	1.032	0.328–3.254			
Matching tooth-wear facets							
No	164 (65.6)		Reference		—	—	—
Yes	84 (33.6)	.57	1.41	0.434–4.586			
Symptomatic TMJ hypermobility							
No	232 (92.8)		Reference		—	—	—
Yes	17 (6.8)	.03	4.757	1.175–19.266			

ORs were calculated relative to the reference value of the variable. Explained variance of the multiple logistic regression model: 8.1%.

participants reporting, or not reporting, intermittent locking suggested no correlation between intermittent locking and these two reported parafunctions.

The univariate logistic regression analysis for opposing matching tooth-wear facets showed that their presence was associated to reported nocturnal tooth grinding (P = .004) and increasing age (P = .01). Tooth grinding remained a significant factor (OR = 2.8, CI 1.4–5.8 , P = .008) in the multiple analysis.

Discussion

In the present study, the hypothesis was tested that oral parafunctions and symptomatic TMJ hypermobility in adolescents increase the risk for both ADDR and reported intermittent locking. The hypothesis was partially rejected because only reported intermittent locking was found to be related to a parafunctional activity, eg, to diurnal jaw clenching.

Table 4 Univariate Logistic Regression Analysis of Risk Factors for Reported Intermittent Locking in at Least One of the TMJs

		Univariate analysis		
Variable	Count (%)	*P*	OR	95% CI
Age (continuous, in years)		.87	0.943	0.458–1.943
Gender				
Boys	4 (17.4)		Reference	
Girls	19 (82.6)	.8	0.722	0.062–8.464
History of trauma				
No	11 (47.8)		Reference	
Yes	12 (52.2)	.15	4.167	0.607–28.621
Reported parafunctions				
Nocturnal tooth grinding				
No	20 (87)	—	—	—
Yes	3 (13)			
Diurnal clenching				
No	12 (52.2)		Reference	
Yes	11 (47.8)	.05	10.02	1.011–104.48
Gum chewing				
No	1 (4.3)	—	—	—
Yes	22 (95.7)			
Nail-biting				
No	6 (21.6)		Reference	
Yes	17 (73.9)	.2	3.25	0.461–22.927
Lip- and/or cheek-biting				
No	11 (47.8)		Reference	
Yes	12 (52.2)	.56	1.714	0.29–10.303
Biting on objects				
No	16 (69.6)		Reference	
Yes	7 (30.4)	.89	1.136	0.16–7.995
Matching tooth-wear facets				
No	15 (65.2)		Reference	
Yes	8 (34.8)	.67	0.667	0.106–4.206
Symptomatic TMJ hypermobility				
No	19 (82.6)		Reference	
Yes	4 (17.4)	.36	0.357	0.039–3.256

ORs were calculated relative to the reference value of the variable. Explained variance of the univariate logistic regression model for diurnal clenching: 27.3%.

The overall prevalence of ADDR in the current adolescent sample was lower than previously reported.[1] This discrepancy could be related to differences in diagnostic techniques (eg, palpation versus palpation and auscultation in the former study) or differences in study samples (eg, smaller sample size, lower socioeconomic status, and higher numbers of nonCaucasian participants in the current sample compared to that in the former study), although race was previously found to be unrelated to ADDR.[1]

The multiple logistic regression analysis for ADDR showed that this condition was related to an increasing age in adolescence, which corroborates earlier results.[1,16] This finding supports the concept that changes in intra-articular spatial relations in adolescence may induce an ADDR.[21] Since joint morphology and intra-articular spatial dimensions

change in adolescence,[22,23] space insufficiency within the TMJ may develop, leading to a disc positional change. On the other hand, ADDR was not found to be related to symptomatic TMJ hypermobility, defined and diagnosed on the basis of the jerky jaw movements arising from the interference of the articular eminence with condylar movement.[18,19] Several previous studies did find a relationship between ADDR and condylar hypermobility, defined as condylar translation beyond the eminence at maximum mouth opening.[24,25] Such condylar translation, however, is observed not only in subjects with symptomatic TMJ hypermobility, but also in up to 80% of those without it.[26,27] This indicates that such condylar translation merely reflects physiologic condylar mobility. Therefore, it was not the condylar translation beyond the eminence, but rather the jerky jaw movements during such condylar translation that were used in the diagnosis of a hypermobility derangement of the TMJ. In fact, the term "TMJ hypermobility" is inappropriate because no excessive mobility occurs in those TMJs. Therefore, it is suggested that instead of "TMJ hypermobility," the term "condyle-eminence interference" be used as it better reflects the character of this functional TMJ disorder.

Intermittent locking was reported by 6 adolescents diagnosed with an ADDR (ie, 46.2% of the ADDR subjects, or 2.3% of the study sample). Since reported intermittent locking is predictive of ADDWoR development,[4,7] this suggests that these adolescents may be at higher risk of permanently losing their joint's reducing ability. This may already have happened in those participants in the sample who reported to have experienced locking but were asymptomatic at the time of examination (ie, without having an ADDR or a limited mouth opening). An ADDWoR is often free of clinical symptoms, such as a movement limitation and pain.[7,28] That 3.9% of the participants in the sample (ie, 10 subjects) may have an ADDWoR may be in line with a previously reported ADDWoR prevalence of 8% in an adult nonpatient sample as detected on magnetic resonance imaging (MRI).[29] Although an ADDWoR is often asymptomatic, its possible long-term consequences in young subjects, eg, an impaired mandibular growth,[30,31] remain to be determined.

While oral parafunctions were not found to be risk factors for ADDR, reported diurnal jaw clenching was found to be a risk factor for reported intermittent locking. This suggests that parafunctional loading may contribute to the temporary loss of reduction of an anteriorly displaced disc, while having no significant role in ADDR onset. When the articular disc is properly interposed, its intermediate zone can sustain heavy loading.[32] If intra-articular changes during growth in adolescence have lead to an ADDR, loading upon the posterior aspect of the disc and/or its bilaminar zone, both structures with no load-bearing function,[33] may influence the disc reduction. The effect of loading was demonstrated recently in an experimental study in which subjects with ADDR who reported intermittent locking were found prone to temporarily lose their reducing capacity after an intensive chewing exercise.[6] An increased intra-articular friction resulting from the loading,[34] or a hyperactivity of the lateral pterygoid muscle,[35] were suggested as possible mechanisms to prevent the anteriorly displaced disc from reducing.

The "gold standard" for the assessment of nocturnal tooth grinding is a recording of the nocturnal masticatory muscle activity by means of polysomnography.[36] However, as this technique is costly and has low availability, nocturnal grinding is usually assessed on the basis of self-report or by the presence of clinically documented tooth-wear facets.[37,38] Tooth wear has a discriminative capacity between young adult sleep bruxers and control subjects.[39] The adolescents' anamnestic report of grinding in the present study was found to be significantly correlated to the presence of matching tooth-wear facets. In combination with the finding of Abe et al,[39] this suggests that selfreport of grinding may be considered an indicator for nocturnal tooth grinding.

ADDR and symptomatic TMJ hypermobility were diagnosed using clinical criteria, which have shown a good reliability.[19] The criteria have been tested for their concurrent validity in comparison to optoelectronic movement recordings and MRI, and good agreement was found with diagnoses based on movement recordings.[18] The clinically detected ADDR and symptomatic TMJ hypermobility usually agreed with the MRI diagnoses, while MRI often diagnosed those derangements in clinically symptom-free joints. The latter discrepancy could be related, among others, to existence of biological variations in TMJ intra-articular anatomy or to uncertainty in MRI interpretation.[18] Based on these outcomes,[18,19] and because of practical and financial reasons, the use of the clinical criteria seems to be sufficient for establishing diagnoses of TMJ internal derangements in clinical studies with large samples.

Intermittent locking was assessed on the basis of self-report, which is inevitably subjective. Therefore, it cannot be excluded that mechanisms other than a temporarily nonreducing disc could have been responsible for the reported movement limitations, eg, fear of movement or muscle pain. However, these mechanisms are not likely to have been involved because the onset and duration of the limitation due to

fear of movement or muscle pain would have been different from those due to intermittent locking (viz, a gradual and persistent versus a sudden and transient limitation, respectively). Moreover, positive reports of movement limitations due to pain only (hence, without the feeling of a locked jaw) were not regarded as intermittent locking to avoid inclusion of myogenous limited mouth openings. Currently, the use of self-report of a significant mouth-opening limitation is generally accepted, even mandatory in the clinical diagnosis of an ADDWoR.[3,20]

This study has determined risk factors for ADDR and for intermittent locking. Only the adolescents' awareness of the presence or absence of parafunctions was noted and not their severity and duration in relation to ADDR and intermittent locking. These features should be taken into account in future studies to assess possible dose-response relationships. However, as participants may be unable to assess the severity of some parafunctions (eg, grinding and clenching), measurements of masticatory muscle activity (eg, polysomnography, electromyography) may be necessary. Likewise, a technique such as the so-called ecological momentary assessment[40] may be needed to obtain an estimate of the parafunction duration in relation to ADDR and intermittent locking.

Another study limitation was the relatively small number of subjects found with an ADDR and intermittent locking, which allowed performing univariate logistic regression analyses with a sufficient power but limited the power of the multiple analysis for ADDR. While ADDR is associated with increasing age, its lack of association with symptomatic TMJ hypermobility could have been due to the low number of ADDR cases. It is unlikely that limited power is responsible for the lack of association between ADDR and parafunction because the different parafunction forms did not appear to be risk factors for ADDR already at the univariate level. Furthermore, the explained variance of the multiple logistic regression models was low (8.1% for ADDR and 27.3% for reported intermittent locking), implying that these conditions have other much more important risk factors. In fact, the low overall model contribution suggests that the diurnal clenching (the weaker factor) might even be absent in any final model that considers stronger factors that were absent in this analysis. Therefore, other factors that lead to ADDR and intermittent locking need to be studied, preferably in larger samples and in a longitudinal design, allowing temporal associations and increasing the probability of finding cause-and-effect relationships.

Within the limitations of this study, it is concluded that, in adolescence, increasing age increases the risk of developing an ADDR, while diurnal jaw clenching increases the risk of intermittent locking. Symptomatic TMJ hypermobility seemed not to be a risk factor for either ADDR or intermittent locking.

Acknowledgments

This study was supported by the Netherlands Institute of Dental Sciences (IOT). Drs W. E. van Amerongen and H. C. Euwe from ACTA, section Pedodontology; Dr C. M. Visscher, Department Oral Kinesiology; and dental students C. Borsje and C. M. C. Volgenant are gratefully acknowledged for their support of this study.

References

1. Huddleston Slater JJ, Lobbezoo F, Onland-Moret NC, Naeije M. Anterior disc displacement with reduction and symptomatic hypermobility in the human temporomandibular joint: Prevalence rates and risk factors in children and teenagers. J Orofac Pain 2007;21:55–62.
2. Farrar WB, McCarty WL. A Clinical Outline of Temporomandibular Joint Diagnosis and Treatment. Montgomery, AL: Normandie, 1982.
3. De Leeuw. Orofacial Pain: Guidelines for Assessment, Diagnosis and Management. Chicago: Quintessence, 2008.
4. Westesson PL, Lundh H. Arthrographic and clinical characteristics of patients with disk displacement who progressed to closed lock during a 6-month period. Oral Surg Oral Med Oral Pathol 1989;67:654–657.
5. Nitzan DW, Dolwick MF. An alternative explanation for the genesis of closed-lock symptoms in the internal derangement process. J Oral Maxillofac Surg 1991;49:810–815.
6. Kalaykova S, Lobbezoo F, Naeije M. Effect of chewing upon disc reduction in the temporomandibular joint. J Orofac Pain 2011;25:49–55.
7. Kalaykova S, Lobbezoo F, Naeije M. Two-year natural course of anterior disc displacement with reduction. J Orofac Pain 2010;24:373–378.
8. Kurita K, Westesson PL, Yuasa H, Toyama M, Machida J, Ogi N. Natural course of untreated symptomatic temporomandibular joint disc displacement without reduction. J Dent Res 1998;77:361–365.
9. Sato S, Sakamoto M, Kawamura H, Motegi K. Long-term changes in clinical signs and symptoms and disc position and morphology in patients with nonreducing disc displacement in the temporomandibular joint. J Oral Maxillofac Surg 1999;57:23–29.
10. Stegenga B, de Bont LGM. TMJ disc derangements. In: Laskin DM, Greene CS, Hylander WL (eds). Temporomandibular Disorders: An Evidence-based Approach to Diagnosis and Treatment. Chicago: Quintessence, 2006:125–136.
11. Magnusson T, Egermark I, Carlsson GE. A prospective investigation over two decades on signs and symptoms of temporomandibular disorders and associated variables. A final summary. Acta Odontol Scand 2005;63:99–109.
12. Baba K, Haketa T, Sasaki Y, Ohyama T, Clark GT. Association between masseter muscle activity levels recorded during sleep and signs and symptoms of temporomandibular disorders in healthy young adults. J Orofac Pain 2005;19:226–231.

13. Nagamatsu-Sakaguchi C, Minakuchi H, Clark GT, Kuboki T. Relationship between the frequency of sleep bruxism and the prevalence of signs and symptoms of temporomandibular disorders in an adolescent population. Int J Prosthodont 2008;21:292–298.
14. Naeije M, Kalaykova S, Visscher CM, Lobbezoo F. Focus article: Evaluation of the Research Diagnostic Criteria for Temporomandibular Disorders for the recognition of an anterior disc displacement with reduction. J Orofac Pain 2009;23:303–311.
15. Hirsch C, John MT, Stang A. Association between generalized joint hypermobility and signs and diagnoses of temporomandibular disorders. Eur J Oral Sci 2008;116:525–530.
16. Isberg A, Hägglund M, Paesani D. The effect of age and gender on the onset of symptomatic temporomandibular joint disk displacement. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1998;85:252–257.
17. Johansson A, Johansson AK, Omar R, Carlsson GE. Rehabilitation of the worn dentition. J Oral Rehabil 2008;35: 548–566.
18. Huddleston Slater JJ, Lobbezoo F, Chen YJ, Naeije M. A comparative study between clinical and instrumental methods for the recognition of internal derangements with a clicking sound on condylar movement. J Orofac Pain 2004; 18:138–147.
19. Huddleston Slater JJ, Lobbezoo F, Van Selms MK, Naeije M. Recognition of internal derangements. J Oral Rehabil 2004;31:851–854.
20. Dworkin SF, LeResche L. Research diagnostic criteria for temporomandibular disorders: Review, criteria, examinations and specifications, critique. J Craniomandib Disord 1992;6:301–355.
21. Huddleston Slater JJ, Lobbezoo F, Naeije M. Mandibular movement characteristics of an anterior disc displacement with reduction. J Orofac Pain 2002;16:135–142.
22. Dibbets JM, Dijkman GE. The postnatal development of the temporal part of the human temporomandibular joint. A quantitative study on skulls. Ann Anat 1997;179:569–572.
23. Katsavrias EG. Changes in articular eminence inclination during the craniofacial growth period. Angle Orthod 2002;72:258–264.
24. Katzberg RW, Keith DA, Guralnick WC, ten Eick WR. Correlation of condylar mobility and arthrotomography in patients with internal derangements of the temporomandibular joint. Oral Surg Oral Med Oral Pathol 1982;54:622–627.
25. Johansson AS, Isberg A. The anterosuperior insertion of the temporomandibular joint capsule and condylar mobility in joints with and without internal derangement: A double-contrast arthrotomographic investigation. J Oral Maxillofac Surg 1991;49:1142–1148.
26. Obwegeser HL, Farmand M, Al-Majali F, Engelke W. Findings of mandibular movement and the position of the mandibular condyles during maximal mouth opening. Oral Surg Oral Med Oral Pathol 1987;63:517–525.
27. Kalaykova S, Naeije M, Huddleston Slater JJ, Lobbezoo F. Is condylar position a predictor for functional signs of TMJ hypermobility? J Oral Rehabil 2006;33:349–355.
28. Emshoff R, Rudisch A, Innerhofer K, Brandlmaier I, Moschen I, Bertram S. Magnetic resonance imaging findings of internal derangement in temporomandibular joints without a clinical diagnosis of temporomandibular disorder. J Oral Rehabil 2002;29:516–522.
29. Bernhardt O, Biffar R, Kocher T, Meyer G. Prevalence and clinical signs of degenerative temporomandibular joint changes validated by magnetic resonance imaging in a non-patient group. Ann Anat 2007;189:342–346.
30. Gidarakou IK, Tallents RH, Kyrkanides S, Stein S, Moss ME. Comparison of skeletal and dental morphology in asymptomatic volunteers and symptomatic patients with bilateral disk displacement without reduction. Angle Orthod 2004;74:684–690.
31. Bryndahl F, Eriksson L, Legrell PE, Isberg A. Bilateral TMJ disk displacement induces mandibular retrognathia. J Dent Res 2006;85:1118–1123.
32. Tanaka E, van Eijden T. Biomechanical behavior of the temporomandibular joint disc. Crit Rev Oral Biol Med 2003; 14:138–150.
33. Hirose M, Tanaka E, Tanaka M, et al. Three-dimensional finite-element model of the human temporomandibular joint disc during prolonged clenching. Eur J Oral Sci 2006;114:441–448.
34. Nitzan DW. "Friction and adhesive forces"—Possible underlying causes for temporomandibulair joint internal derangement. Cells Tissues Organs 2003;174:6–16.
35. Tanaka E, Hirose M, Inubushi T, et al. Effect of hyperactivity of the lateral pterygoid muscle on the temporomandibulair joint disk. J Biomech Eng 2007;129:890–897.
36. Lavigne GJ, Khoury S, Abe S, Yamaguchi T, Raphael K. Bruxism physiology and pathology: An overview for clinicians. J Oral Rehabil 2008;35:476–494.
37. Könönen M, Klemetti E, Waltimo A, et al. Tooth wear in maxillary anterior teeth from 14 to 23 years of age. Acta Odontol Scand 2006;64:55–58.
38. Hirsch C. No increased risk of temporomandibular disorders and bruxism in children and adolescents during orthodontic therapy. J Orofac Orthop 2009;70:39–50.
39. Abe S, Yamaguchi T, Rompré PH, De Grandmont P, Chen YJ, Lavigne GJ. Tooth wear in young subjects: A discriminator between sleep bruxers and controls? Int J Prosthodont 2009;22:342–350.
40. Shiffman S, Stone AA, Hufford MR. Ecological momentary assessment. Annu Rev Clin Psychol 2008;4:1–32.